

***Sheet no.:23***

***Refer to slide no. : 23***

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Please refer to the slides; this sheet contains ONLY extra information that were mentioned.

Slide #2:

-Present diagnostic problems to the clinician: diagnosis of theses acute conditions is hard, your head will be revising all situations related and won’t be able to know from where to start. But for an easier way to diagnose them, you need to have a systematic way of thinking.

Slide #4:

Acute conditions can be one of those mentioned in the slides, in addition to some conditions we took in oral medicine that will be discussed.

Slide #5:

**1-Traumatic lesions**

The photo shows recession, abrasion.

Why did we call it an acute lesion not a chronic periodontitis? Look at the teeth, they look shiny, which means oral hygiene is good, no signs of inflammation and there was no bleeding on probing. Also, look at the papillas, they are located in their normal positions, recession is buccaly only. In this case, it is caused by traumatic brushing.

Why did we call it “acute”? Because the patient might not notice this as a problem right from the beginning, and when you see it the patient will not be able to precisely tell you when it has appeared or give you a proper history, so we will consider it as an acute lesion.

Slide#6:

The photo on the right: shows keratotic areas due to friction ( a traumatic lesion) but without recession because here the gingiva is thick, so over growth and keratosis happened in response to the trauma.

The photo on the left: the doctor advice you not to ever give a spot diagnosis, even if you are seeing caries don’t say it is caries. Be systematic in any oral exam in general, look at the surroundings, ask about the history… . This is an intraoral photograph showing lower premolar areas with canines, showing the labial surface of teeth and an ulceration that is irregular and large in size, it is related to the lower premolar.

-Could it be an aphthous ulcer? No, aphthous ulcers are regular, and we can ask for more information like is it recurrent? Has it been in other areas?

Slide#7:

Physical trauma

Ulcerated lesion showing a part of the root. If you see a patient like this, ask him about habits, in this case the patient uses his nails or the pen cap as a habit and keeps rubbing this area when he is stressed.

Slide#8:

Thermal trauma.

It is a red lesion. Usually you will think about things more related to bleeding or hematoma, but it may also resembles a burn, just ask the patient, you should ask the patient, did you drink something hot recently? For example, if the answer was yes we will simply reach the diagnosis “thermal trauma” by taking a proper history.

Slide#9:

This is a chemical burn with hydrogen peroxide.

Slide#10:

After removing the cause, if lesions are symptomatic> give pain killers. Or give him mouthwashes to prevent infection.

Orabase: they are given to cover the lesion, act as covering base so the lesion won’t hurt anymore.

They used to use الطحينية as an orabase.

Slide#11:

**2-Bacterial lesions**

Slide#12: the same

Slide#13:

Necrotizing ulcerative gingivitis: the used to call it “Trench mouth disease”.

Trench : خندق, it was then epidemic in wars, soldiers were stressed “which means they had low immunity “.

Vincent’s gingivitis: named after the bacteria that cause it ”!”.

Other species like spirochete which are motile bacteria.

The photo shows a very bad oral hygiene, plaque is everywhere, a white layer is present and there are signs of spontaneous bleeding and severely inflamed gingiva. In severe gingivitis you might have spontaneous bleeding, but there is a psuodomembrane (necrosis of epithelial tissue). So, the characteristic signs of this disease are:

Slide#14:

M>F “it’s written in the slides M=F” , adolescents not children, more common in college student “the doctor encounters almost every year a dental student that have NUG, not so severe but the student usually complains of localized pain and that the papilla are no longer there in a specific tooth. It is usually related to stress”.

Why NUG happened and not pocketing? Because stress means low immunity.

Slide#15: the same.

Slide#16:

Features:

-pain: it is one of the very few issues in perio that might cause very severe excruciating pain, neither gingivitis nor periodontitis do. Pockets cause discomfort only, not pain.

-psuodomembrane is a sign but not really a main one, because the patient might have wiped it.

-Flat or reversed margins because of necrosis that happened to the tips of papillas. Reversed margins means buccaly higher than proximally because usually it starts interdentally then goes to gingival margins.

-bad odour, it smells like الكبريت. Pasty saliva means thick and frothy.

These are the main three signs and the other might follow.

Slide#17:

The photo shows the disease in its early stages.

The pain comes from the necrosis.

-When will it be pocketing and when it will be necrosis? In periodontitis it is a chronic disease the immunity is high so the junctional epithelium and bone starts to detach and go away to protect themselves from the bacteria and pockets result “as an immune response”, but here, instead, the immunity suddenly becomes low in addition to the presence of other predisposing factors, necrosis happens to the junctional epithelium, the CT and the outer epithelium, so all the gingiva is necrotized, that’s why it’s extremely painful.

Slide#19: papillas are flat.

Slide#20:

So, if you saw something similar like that, always ask about the history, social history is important as well as medical history (possibility of having hepatitis B or HIV).

Biopsy is not really indicated as a diagnostic investigation, because it will show a typical necrosis.

There is a case study in the diagnosis clinics, a 19 yrs old patient presented with gingival swelling, it was thought to be a classic case of NUG, but it wasn’t, it was painless and interdental papillas are just swollen, they are not absent, molar areas where covered with gingival tissue with peripheral necrosis and pseudomembrane (the only sign related). They asked for CBC, it turned out that the patient has leukaemia.

So always try to review history with what you know clinically.

Slide#21:

-acute episodes (like pain and severe inflammation) usually resolve, but the problem is in the recurrence.

It might also progress into necrotizing ulcerative periodontitis (NUP) if not treated, exceeding the gingiva downward toward attachments and bone and even toward the mucosa if not treated resulting in necrotizing ulcerative stomatitis.

-when it heals by itself “without treatment” fibrous scarring results, the gingiva becomes thick and the architecture looks reversed. The treatment include gingivoplasty to reshape the contour.

 Slide#22:

This photo shows localized NUP, necrosis went down toward the attachment, bone and PDL.

Necrosis of bone ends with sequestrum ( part of the dead bone comes out), just like in the photo in Slide#24.

Slide#25:

This is a healed case; papillas have reversed architecture, and sever bone loss as it appears in the radiograph.

Slide#26:

Necrotizing ulcerative stomatitis, it happens when it exceeds the alveolar bone toward the mucosa. We can clearly see the bone sequestrum; sometimes it may be complicated with oro-antral fistula.

If it progresses more, it might result in Cancrum Oris or Norma, especially in immuncompremised patients (malnourished people like in Africa). So, it progresses to the periodontium then the mouth then outside the mouth, and this is a fatal condition, luckily it doesn’t get that bad in our countries, we don’t get malnourished.

Slide#27:

When you see this photo you might think of periodontitis with recession, but here the papillas have lost their architecture (not even scalloped). On the right, the photo shows the case after treatment, scar tissue and thick gingiva, it might need gingivoplasty later on (an attempt to get the contour back).

Slide#28:

We should do full treatment, but you should firstly alleviate the symptoms.

We have to know the cause, is it smoking? Lack of sleep?

But bacteria is always a cause, when a patient comes, try to wipe the area with a warm saline or with hydrogen peroxide (it extracts oxygen and kills anaerobic bacteria), and try to expose the tooth. Scaling in this stage is so painful so we don’t do it.

Slide#28:

In the left photo, u may think that its periodontitis with recession , actually it can be, but here you notice that this case has no papilla “lack papilla”.. but if we have periodontitis and gingival recession we will still have papilla architecture .

After treatment you notice healed gingiva have scar tissue, here we may need gingivoplasty later on

Slide#29-30:

You should do full treatment for the patient.

You should try to reduce the symptoms. (pain >> give analgesics)

You have to know the reason why we have the disease ( smoking, lack of sleep ..) but also plaque is the reason , there is a bacteria that causes the problems .

In the past they used to give AB for every patient with NUG , but now when u have a patient with NUG at first try to get wet gauze with warm saline or hydrogen peroxide “it releases oxygen >> kills anaerobic bacteria” and it will do some wiping of dead tissues

At this point you don’t do scaling , its very painfull

No brushing at least the first 1-2 days.

Slide#31:

2nd visit you do only supragingival scaling.

Slide 32-33:

If we have disseminated infection we usually give metronidazole, our aim is to give high dose for short duration, but 250 mg for 5 days is usually enough

Systemic conditions it they have any need to be controlled.

Slide#34:

Maintenance: if we have a case with high tendency of recurrence, we bring the patient back to see what happened with him

Slide#35:

In case of no- responsive site, you have to look for your diagnosis, your treatment.

If the cause was stress you can send the pt to counseling.

Slide#36-37:

Better architecture after treatment.

Slide#38-40:

The second topic in this lecture is about abscesses.

What is abscess? Collection of pus

What is pus? Bacteria, RBCs, but mainly WBCs

Types of abscess? Periapical (acute or chronic), pericoronal = pericoronitis, periodontal abscess is of 2 types (gingival and periodontal)

Slide#41-42:

Intra-oral photograph showing swelling buccal to the canine

First thing to do for diagnosis is to ask about history, when it appeared? “after restoration, after severe pain, or eating something”

Irrigation can be done as a treatment for gingival abscess

So the patient don’t have periodontitis, but in the history u will notice he had eaten sth before the swelling appear >> gingival abscess

Slide#43-44:

Periodontal abscess is the second dental emergency after periapical abscess, it’s common.

Why abscess happens in general? Why it happens in endo?

Why we have more severe pain in pulps? It’s a closed chamber that cannot increase in size upon inflammation>> it will generate pressure.

When this inflammation get out periapical lesion/ radiolucency will initiate and enlarge, if it continue to enlarge without drainage , it will cause severe pain , but sometimes (if the bone was thin) then it will open into an alveolar sinus >>drainage . [sometimes when you open an access pus will get out from the canal if it didn’t find any way through tissues ]

Periodontal abscess usually occur in patients having periodontitis, due to blockage of the socket.

In periodontitis the drainage of pus, inflammation and infection occur through the socket , if blockage happened at some point>>periodontal abscess .

Causes of pocket blockage?

1. deep pocket,
2. irregular pocket that facilitate calculus accumulation at the upper aspect of the pocket and blockage
3. after treatment, due to inadequate irrigation during treatment that leads to stuck in of calculus and blockage, or because you only clean the upper part of the pocket if its deep “unexperienced doctor”
4. Diabetic patient with poor control and have periodontitis, you can detect those patients by the number of abscesses they have (multiple abscess /3,4,5,6…)
5. Patients with periodontitis and pus where given AB >> super infection >>abscess. because u didn’t remove the cause of the disease [some dr.s know nth about health and perio,,whenever they saw infection they give AB ]

So AB without treatment can cause abscesses.

Slide#45:

\*\* you have to study the tables well\*\*

Slide#46:

The site of the periodontal abscess usually differs from that of periapical abscess “at the apex” but not always.

Which of them is more tender to percussion? Periapical abscess, but periodontal abscess can be tender if the pocket is down. “I think he means its v. deep”

What about vitality test? Periodontal abscess is more vital , it’s the best test to do

Presence of periodontitis , or history of periodontal treatment before the appearance of the abscess can help to differentiate , or if the pt had new restoration , or incomplete endo treatment, large filling or large cavity help in diagnosis.

Examination: Usually in periodontitis inflammation is generalized.

Radiograph we can see the position but it’s not v. accurate .

So we have clinical examination, history, vitality, percussion, and radiograph.

Slide#47:

Abscess that heals perfectly after periodontal treatment.

Slide#49:

Here we have severe abscess at the site of the furcation (which is really difficult to clean), even if you do incision and drainage it may not heal. So if it’s not accessible area you raise a flap, clean it and close the flap.

If you are not competent to raise a flap>> you follow the cause, you may do endo (not from the first day), but usually we do incision and drainage then do irrigation, then you follow the pt to remove the etiology (RCT for ex.)

In periodontal abscess it’s the same but the incision is usually done in the pocket to open the sulcus. if it’s a really large rounded abscess you may do the usual incision.

5 main scenarios to give Abx : aggressive periodontitis, severe non-responsive periodontitis /refractory periodontitis )here you may take a sample from the pocket to know the type of the bacteria presented and give AB accordingly(, necrotizing in case of systemic involvement, abscesses if it involve dissemination or systemic condition , and immune-compromised patient.

Slide#54:

Surgical flap elevation demonstrates calculus also.

Slide#56:

Multiple abscesses with multiple fistulas. Here the patient has poor oral hygiene so DM is not the only cause.

Slide#60:

Radiographic tracing, the tooth has no cavity or restoration >> periodontal abscess .

Slide#63:

This is Flap.

Slide#67:

What determines the prognosis of the tooth is the attachment.

Periapical abscess: healing and success of the treatment is high, no loss of attachment [tooth is not considered hopeless]

Periodontal abscess: prognosis in not great but you can still save the tooth, there’e pocketing and attachment loss.

If the tooth has periodontal abscess twice >> extract it.

Slide#68:

Abscess at the lower anterior area, it was perfectly healed after treatment , but the patient had slight recession. On radiograph we can see that only 10% of the tooth is attached, poor prognosis, maybe hopeless. after 6 months the patient had trauma on it >> the tooth get out !!!

Slide#69:

1. Multiple adscesses
2. Tracing

Here we have lost the attachment on one wall only, the other wall have bone but with different radiolucency (the left side)

We have to do vitality testing , probing (look for pockets), clinical testing and history to know what the tooth needs exactly.

يا اخوان هذه الشيت تحتوي على المعلومات الإضافية فقط, اذا درستها لحالها لازم ضميرك يأنبك, واذا تريدون الدراسة من مصدر واحد فقط فالسلايدز أولى.

**بالتوفيق :))**